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Plant Thermomorphogenesis:
Identifying Plant Thermosensors

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Abstract
Plants are subjugated to fluctuations in temperature over daily and seasonal temporal scales. Because of their sessile nature, plants have evolved necessary physiological response pathways to temperature fluctuations in order to adapt to their environments. There is an abundance of research covering temperature stress response, but plant response to non-stressful temperature fluctuations has closer applications to fields such as climate change, due to the incremental shifts in temperature caused by the buildup of greenhouse gases. Temperature shifts due to global warming and normal seasonal variations have important effects on the distribution of plant species and the effect on crop plant supply. Due to this significance, this review will highlight current understandings of the mechanisms that control plant thermomorphogenesis, present novel findings in plant thermosensors, as well as investigate other areas of research that are necessary to further understand the mechanisms of plant adaptation to ambient temperatures. For literature outside the scope of this review, readers are referred to [1].

Introduction

The term "thermomorphogenesis" was coined in 1989 by Erwin and colleagues to describe the effects of temperature on plant morphology [2]. Erwin's usage of the term was in comparison to photomorphogenesis, and used to describe the impact of the difference in day-night temperatures on stem elongation and leaf orientation in *Lilium longiflorum* [2]. In this review, the term will be used to refer to plant morphological changes that allow the plant to adapt to temperature shifts that may otherwise be detrimental to either plant growth or development.

Thermomorphogenesis relies on plant thermosensors, a term which has been generally used to describe plant components involved in sensing temperature. However, recent work has argued for a narrower definition of the term. Vu and colleagues argue that in sensory systems, sensors must decode a signal through an alteration of structure, activity, or interaction in order to trigger downstream responses [3]. As a result, they have suggested that plant thermosensors be more narrowly defined to three criteria: (i) temperature directly impacts the thermosensor's properties, (ii) the modified properties play an important role in the signal transduction of temperature response, and (iii) these changes lead to relevant changes in plant physiology or morphology [3]. Recent research has identified and investigated more plant thermosensors as a result of this new definition.

Identifying key components of plant thermal response

Cyclic Nucleotide Gated Calcium Channels

Calcium channels in the plasma membrane were among the first components identified to be involved in temperature response in plants [4, 5, 6]. Temperature shifts can be detected by cyclic nucleotide gated ion channels (CNGCs), likely by the increase in fluidity of the membrane, which causes the activation of calcium kinases [7, 8]. In *Arabidopsis*, CNGC2 is an essential

component of thermosensing, and a targeted CNGC2 deletion resulted in the loss of a thermoresponsive calcium channel in the membrane [7]. Similar experiments using Ca2+ channel blockers significantly decreased the heat response which negatively affected acquired thermotolerance [6]. The amplitude of calcium influx within the first few minutes of temperature increase are essential to determine the level of activation of the plant's heat response [6]. Increased deficiency of CNGC2 results in a higher influx of calcium, which resulted in plants with hyper-thermosensitive phenotypes [6].

The activation of calcium-dependent protein kinases (CDPKs) due to the influx of calcium can activate mitogen-activated protein kinases (MAPKs) or NADPH oxidase, which produces reactive oxygen species (ROS) [9]. ROS is significantly involved in plant growth and development, as well as responses to environmental stimuli [9]. A thorough overview of downstream signaling transduction pathways activated by the influx of calcium through CNGCs can be found in Figure 1.

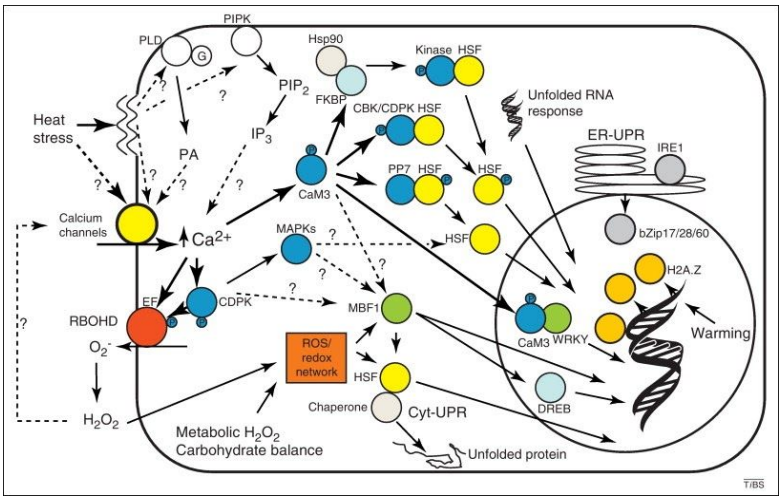

Figure 1: ***Components of heat response inside the cell.*** *Many mechanisms remain unknown, but calcium channels are shown to play a pivotal role in sensing increased temperatures.*

H2A.Z-Containing Nucleosomes

Chromatin-level regulation has also been shown to play a key role in plant thermosensing. H2A.Z-containing nucleosomes participate in the coordination of transcriptome response to ambient temperatures [4]. An increase in temperature results in the decrease of H2A.Z-containing nucleosomes, which wrap DNA more tightly and decreases the ability of RNA Polymerase II to transcribe genes [10].

This decrease in the nucleosomes therefore increases the expression of temperature-responsive genes, such as HSP70 and FLOWERING LOCUS T (FT) [4]. The PHYTOCHROME-INTERACTING FACTOR4 (PIF4) binding site in the FT promoter includes the presence of H2A.Z-nucleosomes, suggesting in the absence of those nucleosomes, PIF4 would be able to bind more strongly to the FT promoter [11, 12].

PIF4 as a Centralized Hub of Temperature Response

Elevated temperatures result in adaptive morphological changes including hypocotyl elongation and early flowering. The transcription factor PIF4 has been identified as a key regulator that works alongside the thermosensor H2A.Z to control plant acclimation to higher temperatures, including such architectural changes [13]. These regulations primarily are enacted through PIF4's binding to promoter sequences, including auxin biosynthesis genes and the FT promoter [12, 14]. These transcriptional changes result in downstream effects, primarily phytohormone-induced elongation responses [15].

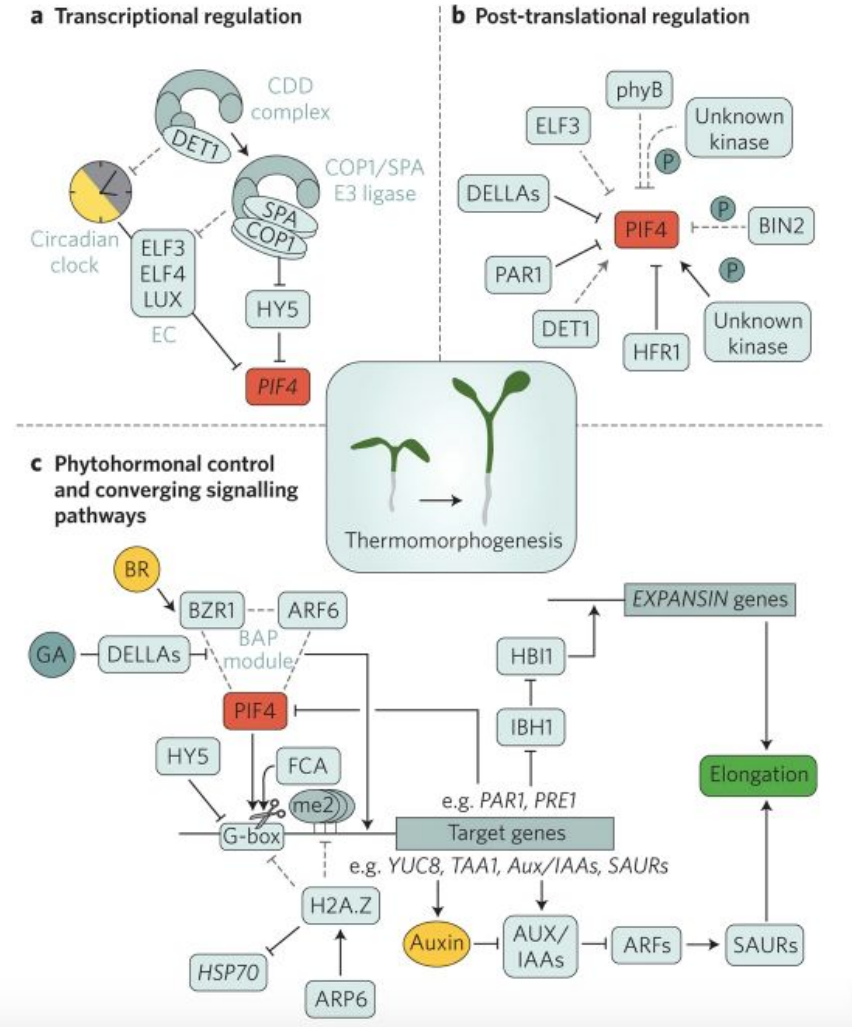

Figure 2: ***PIF4 is a central hub of thermoresponsive growth and development****. These effects are triggered by elevated ambient temperatures and result in thermomorphogenesis, including phenotypic differences such as elongated hypocotyls and early flowering.*

PIF4 is not only involved in transcriptional regulation, but also post-translational regulation and phytohormonal control, each stage of which is tightly coordinated with associated proteins such as BIN2, DELLA proteins, and BZR1, as well as internal mechanisms such as the circadian clock and hormones such as auxin [15]. Figure 2 outlines a simplified model of PIF4's role in thermomorphogenesis.

However, PIF4's activity appears to be conserved regardless of phytochrome and DELLA protein conservation, as shown by the maintenance of hypocotyl elongation in phyBDE-triple mutants and in a DELLA quintuple mutant [16]. Furthermore, transcription of Heat Shock Protein70 (HSP70), a responder to cellular temperature changes, was unaffected by knockout PIF4 mutants [10]. These results suggest that PIF4 does not play a role in temperature sensing, but rather plays an essential part in regulating plant development in response to a rise in ambient temperature. These implications support Vu and colleagues' argument that PIF4 should not be considered a plant thermosensor, as was previously regarded as. According to their narrower definition, PIF4 fails to meet the criterion that temperature directly affects the component's properties such as structure or activity, and therefore should be considered a hub for thermoresponsive growth rather than thermosensory activity [3].

Phytochrome B

On the other hand, temperature shifts do have a direct effect on the conformation of phytochrome B (phyB), a red light receptor. Much research focuses on phyB's role in photomorphogenesis, where red light changes phyB from its inactive P_r to its active P_{fr} state [17]. High temperatures can reverse this transition to inactivate phyB, in a process called thermal reversion. Thermal reversion was once considered relatively slow (and therefore irrelevant) as compared to light reactions, but recent research has revealed that this is not the case. Using in vivo and in vitro spectroscopy, as well as confocal microscopy to analyze phyB nuclear bodies, the activity of phyB was proven to decrease with increased temperatures across a wide range of light conditions [18]. Thermal reversion of phyB releases the repression of PIF4, which results in thermal response as outlined above [19].

These results indicate a combination of light and temperature sensory systems to optimize the plant response to a wide range of ambient environmental conditions [18]. The conformational change in phyB results in signal transduction and temperature-dependent physiological shifts, aligning with Vu and colleagues' definition of plant thermosensors [3].

Plants grown in darkness usually do not elongate due to higher temperatures, so thermomorphogenesis effects have traditionally been ignored in etiolated seedlings. However, recent studies have revealed that high ambient temperatures suppress apical hook formation, especially regarding ethylene-induced hook formation [19]. Elevated temperatures additionally decreased the auxin concentration ratio along the sides of the seedlings, resulting in decreased hook curvatures [19]. This reduction in auxin biosynthesis is required for the antagonism of ethylene-induced exaggerated hook formation, and this phenotype of thermosensing in the darkness is not expressed in phytochrome-PIF4 mutants [19]. Based on these results, thermo-sensing in etiolated seedlings cannot be explained by the usual thermal response components such as PIF4 and phyB that are studied in light-grown plants.

Emerging plant thermosensors following defined criteria

Following the proposed modifications to the criteria that define a plant thermosensor, new components of plant thermomorphogenesis have been identified or proposed to be plant thermosensors. Some of these components are discussed in these brief reviews [20, 21]. While PIF4 cannot be considered a thermosensor following Vu and colleagues definition, it remains a central hub mediating the growth and development of plants under higher ambient temperature conditions. Recent research has therefore focused on the integration of various signaling pathways to regulate the abundance or transcription of PIF4 [22]. Some of these signaling pathways are succinctly depicted in Figure 3 below [22].

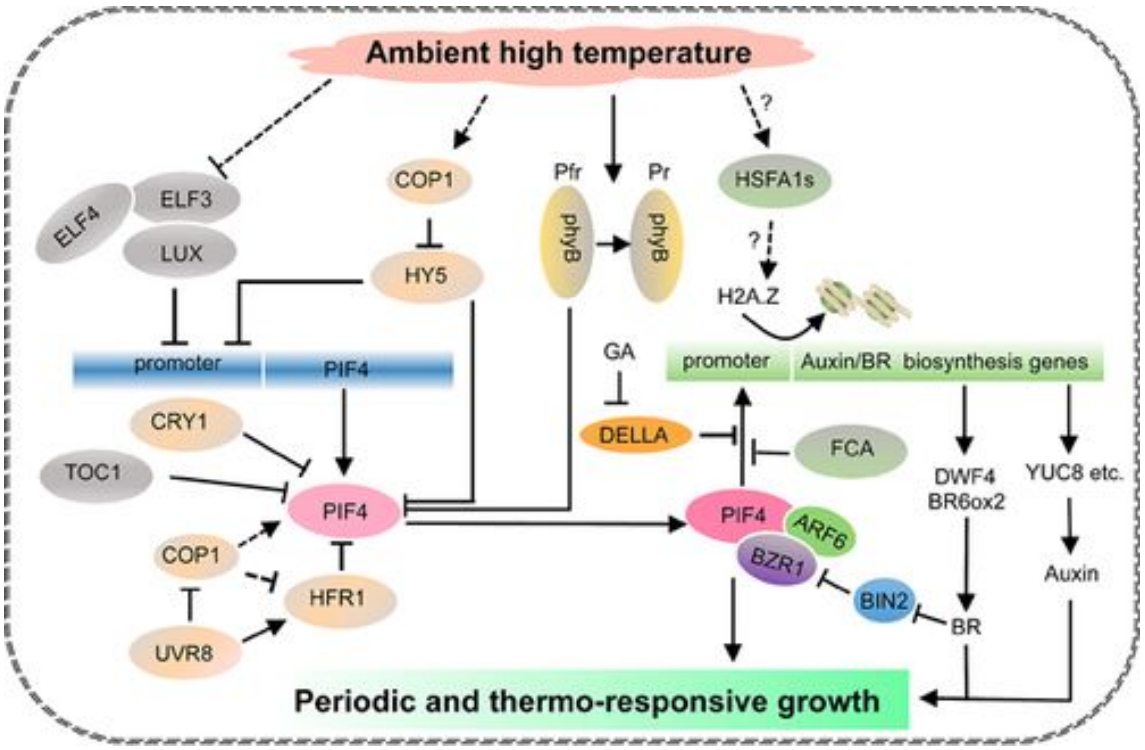

Figure 3: ***Thermosensing pathways for elevated ambient temperatures****. There are four hypothesized pathways for thermosensing which impact downstream plant thermomorphogenesis. The EC and phyB are established thermosensors, but COP1 and HSFA1s require further research. Each of these pathways affect the role of PIF4 as the central hub of thermal response and overall plant thermomorphogenesis.*

RNA-Hairpin Thermoswitch

Recent discoveries of plant thermosensors include a temperature-sensitive molecular RNA translation switch, which was previously studied in bacteria and viruses [20]. Through ribosome profiling, the *Arabidopsis* transcription factor PIF7 has been found to play an important role in plant thermomorphogenesis [23]. To mimic a more realistic natural temperature pattern, Chung et al. studied the effect of elevated temperatures only during midday, as opposed to the traditional continuous elevated ambient temperatures that most plant thermosensory experiments

are subject to [23]. Under these conditions, PIF7 proteins were found to quickly accumulate in response to warm daytime temperatures [23]. PIF7mRNA contains a hairpin structure in the 5'-UTR which forms at low temperatures to block translation of PIF7 [21]. Elevated temperatures directly lead to a conformation in the RNA hairpin, leading to increased protein synthesis and necessary PIF7 control in the expression of key thermomorphogenesis genes, including the auxin biosynthesis gene YUCCA8 [24]. These results indicate the key role of the RNA hairpin thermoswitch in plant thermal sensing and downstream morphological effects such as early flowering and elongation of hypocotyls and petioles [23]. Interestingly, these morphological effects are restricted to daytime and commence rapidly upon exposure to warmer temperatures, with changes in translational efficiency of genes such as PIF7 occurring within a timespan of fifteen minutes [23].

Chung et al. go further to examine whether PIF7 is necessary for thermomorphogenesis, especially given the essential role that PIF4 plays as a central hub of temperature regulation [23]. The effects of PIF7 and PIF4 on hypocotyl elongation appear similar, suggesting control may be redundant, as a *pif4 pif7* double mutant revealed no additive effects [23]. PIF7 is additionally required for petiole elongation and stomatal index, and appears to be particularly relevant for daytime growth under long photoperiods [23].

Evening Complex (EC) Thermosensor

The Evening Complex (EC) is composed of the DNA binding protein LUX ARRHYTHMO (LUX), EARLY FLOWERING 3 (ELF3), and ELF4, and functions as a transcriptional repressor complex and an essential component of the plant circadian clock [25]. ELF3 specifically is important, being both necessary and sufficient to form a complex between ELF4 and LUX, the latter of which targets the EC to the promoter of PIF4 [26]. The EC represses PIF4 transcription and is diurnally regulated, peaking at dusk [26]. Warmer ambient temperatures result in the loss of EC activity, promoting PIF4 expression and leading to plant thermomorphogenesis [25].

Recent structural studies and in vitro assays have furthered understanding of the mechanisms behind EC-DNA binding [25]. While LUX binds to DNA with high affinity alone, the LUX-ELF3 complex is a relatively poor binder, and requires ELF4 to facilitate stronger binding of EC to its DNA target [25]. Warmer temperatures result in weaker binding of the EC to DNA, indicating that ELF4 is a key component of thermosensitive EC activity [25]. This response to warmer temperatures is likely due to the correlation between the length of a polyglutamine (polyQ) repeat, embedded in a prion domain (PrD) in ELF3, with thermal responsiveness [27]. ELF3 has been found to adopt two conformations: an active soluble form at low temperatures, and a higher-order multimeric form at warmer temperatures [27]. Based on electrophoretic mobility shift assays (EMSAs) and in vitro studies, these results suggest that the PrD in ELF3 functions as an environmental switch that can affect EC-DNA binding ability at low vs. high ambient temperatures [27]. This conformational shift allows ELF3 to act as a thermosensor and

quickly and reversibly bind to promoter regions of PIF4 to repress transcription at lower ambient temperatures, while high temperatures result in ELF3's multimeric conformation and prevent its crucial role in the EC's binding ability to DNA [28].

Proposed areas of future research

COP1

High ambient temperatures can trigger the nuclear import of COP1, but this mechanism has not yet been determined [29]. COP1 promotes the expression of PIF4 and increases its stability, while simultaneously targeting HY5 for degradation [30]. Using knockout mutants of COP1 and HY5, the degradation of HY5 by COP1 has been found to be consistent regardless of light responses and timing information [29]. HY5 inhibits thermomorphogenesis by binding to the promoters of PIF4 target genes, competing with PIF4 and preventing thermoresponsive morphological changes such as elongation growth [30]. These results suggest that COP1 plays an essential role in the thermal response pathway, but the exact mechanisms of nuclear import triggered by elevated ambient temperatures are unknown. Further research could reveal whether higher temperatures cause a direct modification to COP1's structure or activity and define COP1 as a potential thermosensor.

HSAF1s

The role of H2A.Z's role in plant thermal response has already been established. However, recent research has revealed that the decrease in H2A.Z-containing nucleosomes from the transcriptome, allowing plant thermomorphogenesis, is dependent on the HSFA1 clade of *Arabidopsis* heat shock factors (HSFs) [31]. Based on ChIP-seq data, immunoprecipitation experiments, and linear models, HSFA1a has been determined to initiate a transcription cascade in response to elevated temperatures, resulting in a decreased H2A.Z-nucleosome signal and increased chromatin accessibility [31]. However, the mechanisms by which elevated temperatures result in HSFA1's initiation of the cascade, as well as the mechanisms by which HSFA1 and H2A.Z nucleosomes influence one another, remain unclear. One recent study has found that there is diurnal variation in thermotolerance in *Arabidopsis*, with miximal thermotolerance during the day [32]. These results suggest that light-dependent pathways may play a role in the HSFA1 transcription cascade, with light signals such as PQ redox state or associated H2O2 generated by chloroplasts diffusing into the nucleus to activate HSFA1s [32]. The presence of HSFA1a around H2A.Z-containing nucleosomes suggests that HSFA1a may recruit chromatin remodelers such as RNA Polymerase II to facilitate H2A.Z eviction, but the exact mechanisms to H2A.Z eviction remain unknown [31]. If this were the case, HSFA1 would not be considered a plant thermosensor, because its role is downstream of the temperature sensory pathway. Increased understanding of the integration of light and temperature perception and response with regards to HSFA1 and H2A.Z would expand our knowledge of plant thermosensors.

CLKs Role in Alternative Splicing

In *Arabidopsis*, 870 genes are alternatively spliced under high temperature, but the mechanisms behind the modulation of alternative splicing by elevated temperatures remains unknown [33]. Lin et al. reference a recent mammalian study to propose a potential thermosensor in the form of CDC-Like Kinases (CLKs) [20]. In the study, physiological temperature changes resulted in structural rearrangements of the kinase activation segment, leading to differential activity levels [34]. Lower body temperatures activated CLKs, which phosphorylate serine/arginine (SR) proteins, key components of the spliceosome [34]. SR proteins control transcription and processing events, from splicing, to nuclear export, translation, and degradation [35]. The mammalian study confirmed that CLKs are key thermosensors that sense physiological temperatures to control alternative splicing and gene expression [34].

Lin et al. highlight the fact that CLKs are highly conserved among mammals and plants, with three homologs in *Arabidopsis* and identical key residues [20]. They also point to previous studies where one *Arabidopsis* CLK directly phosphorylates four SR proteins, suggesting that the mechanisms for thermosensing in mammals already exist in plants [36]. Taken together, these overlapping components suggest that plant CLKs could play a role in thermoregulation and potentially even act as thermosensors themselves to regulate alternative splicing and gene expression. Because this area of research has not been thoroughly explored, further studies are necessary to support or disprove this hypothesis.

HOOKLESS1

HOOKLESS1 (HLS1) has recently been found to positively regulate thermomorphogenesis, largely through its interaction with PIF4 [33]. Based on RNA-seq data, HLS1 and PIF4 have been determined to coregulate the high temperature-response transcriptome, with dependent differentially expressed genes largely overlapping [33]. These results indicate that HLS1 acts synergistically with PIF4 to modulate both transcriptional and posttranscriptional pathways [33].

Elevated temperatures' effect on etiolated seedlings through ethylene-induced hook formation has already been established. Relevantly, *hls1* mutants do not exhibit exaggerated apical hooks when grown under ethylene treatment [37]. Ethylene was found to induce HLS1 transcription, which mediates the auxin distribution ratio that forms the hook in seedlings [38]. Additionally, *hls1* knockout mutants exhibit hyposensitivity to cell elongation and transcriptional changes triggered by elevated temperatures [37]. These results indicate that HLS1 acts as a positive regulator of plant thermomorphogenesis, but further research is needed to determine the exact mechanisms that determine these effects.

Furthermore, HLS1 has been found to play a crucial role in plant defense. *hls1* mutants display enhanced disease symptoms in response to fungal and bacterial infections, accelerated

senescence, and impaired responses to the plant hormone ABA, indicating HLS1's key role in regulating these responses [39]. Interestingly, research suggests that there exists a trade-off between plant growth and defense responses [22]. PIF4 has been found to play a role in down-regulating defense-related genes, with *pif4* mutants more resistant to infection [40]. Based on this research, the interaction between HLS1 and PIF4 could be an important area of research to determine how plant growth and defense responses are balanced, especially in the context of higher ambient temperatures.

Chromatin-Remodeling Processes

Recent studies have revealed the role of the chromatin-modifying enzyme HISTONE DEACETYLASE 9 (HDA9) in evicting the H2A.Z histone variant from YUCCA8 nucleosomes [41]. YUCCA8 is the rate-limiting enzyme in the production of auxin [41]. At warmer ambient temperatures, HDA9 protein levels are high and mediate histone deacetylation, allowing PIF4 to bind to YUCCA8 promoters and increase auxin production, leading to thermomorphogenesis [41]. HDA9 operates at least partially independently of the phyB thermosensory pathway and shade avoidance pathway, but further studies are necessary to determine exactly how HDA9 is directly or indirectly affected by increased temperatures [42]. Of additional interest is whether the effect of HDA9 on H2A.Z eviction extends beyond the YUCCA8 locus.

POWERDRESS (PWR) has also been identified as a protein that interacts with HDA9 [43]. Through transcriptome studies and meta-analysis, mutations in PWR have been found to impede thermomorphogenesis, resulting in global misregulation of genes that are largely overlap with the presence of H2A.Z-containing nucleosomes [43]. PWR and PIF4 are suggested to act in the same genetic cascade for the elongation phenotype, as *pif4 pwr* double mutants were not significantly different from either single mutant [43]. However, the loss of PWR appears to be overcome by PIF4-mediated FT expression for the flowering phenotype [11]. These results indicate that PWR plays a crucial role in developmental processes through its interaction with HDA9, and therefore is of significant interest in ambient-temperature response in plants.

Conclusion

As sessile organisms, plants must modulate their growth and development to respond to fluctuating environmental conditions. The mechanisms of light response, defined by photomorphogenesis, has been widely studied, but temperature sensing and regulation is similarly crucial to plant survival and adaptation. Understanding plant thermomorphogenesis is crucial in the contexts of climate change and food supply. Less studied still is plant responses to ambient temperature increases and the exact mechanisms behind thermosensing and thermoregulation in those temperature ranges. This area of research is extremely relevant to broaden our understanding of plant resiliency in our changing climate, as well as of significant interest due to the application to food supply to meet the accelerated population growth.

Increased ambient temperatures also increase plant respiration rates, which has significant consequences for plant performance and atmospheric CO_2 concentrations in the future [44].

The transcription factor PIF4 acts as a central hub of plant thermal response, including early flowering and hypocotyl and petiole elongation. Most research focuses on the downstream effects of PIF4, but fails to account for how PIF4 is activated and regulated by plant thermosensors that detect high ambient temperatures in the first place. Recently, attention has been drawn to plant thermosensors through Vu and colleagues work in compiling a review of current research and redefining plant thermosensors to meet certain criteria. Specifically, their narrowing of the definition excluded PIF4 as a plant thermosensor due to the lack of a direct change on PIF4 by elevated temperatures [3]. This work has led to a broader search for the exact mechanisms underlying temperature sensing in plants, and led to the discovery of the RNA-hairpin thermoswitch and the EC thermosensor. General understanding of thermosensing prompts further exploration of the role of COP1, HSAF1s, CLKs, HLS, and chromatin-remodeling mechanisms in plant thermomorphogenesis. Furthermore, sensory pathways do not exist in isolation, so it is of significant interest to determine how light signaling pathways, shade avoidance, the flowering pathway, and defense mechanisms interact with temperature sensing and regulation. Clearly, much more research is necessary to fully understand how increased ambient temperature impacts plant morphology and development through various signaling pathways. These discoveries of plant thermosensors will allow future researchers to investigate evolutionary traits driven by temperature across seasonal and geographical scales.

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